WEST Search History

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DATE: Monday, December 05, 2005

Hide?	Set Name	Query	Hit Count
		DB=USPT; PLUR=YES; OP=OR	
☐	L10	kinesin with inhibitor	26
		DB=PGPB,USPT; PLUR=YES; OP=OR	
☐	L9	kinesin with inhibitor	101
☐	L8	kinesin same inhibitor	263
☐	L7	kinesin and l5	0
☐	L6	microtubule and L5	1
☐	L5	4,391,904.pn. or 5,143,854.pn. or 5,559,410.pn. or 5,585,639.pn. or 5,576,220.pn. or 5,541,061.pn. or 3,817,837.pn. or 3,850,752.pn. or 3,939,350.pn. or 3,996,345.pn. or 4,277,437.pn. or 4,275,149.pn. or 4,366,241.pn. or 5,644,048.pn. or 5,386,023.pn. or 5,637,684.pn. or 5,602,240.pn. or 5,216,141.pn. or 4,469,863.pn. or 5,235,033.pn. or 5,034,506.pn. or 5,010,175.pn. or 5,288,514.pn. or 5,539,083.pn. or 5,593,853.pn. or 5,569,588.pn. or 5,549,974.pn. or 5,525,735.pn. or 5,519,134.pn. or 5,506,337.pn.	30
☐	L4	5527773.pn.	1
☐	L3	(microtubule same motility same inhibit$ same ATPase)	16
☐	L2	((microtubule same motility same inhibit$ same ATPase) AND @pd>20051205)	0
		DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR	
☐	L1	(microtubule same motility same inhibit$ same ATPase) AND @pd>20051205	0

END OF SEARCH HISTORY

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FILE 'HOME' ENTERED AT 14:58:48 ON 05 DEC 2005

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FULL ESTIMATED COST	0.21	0.21

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	0.27

FILE 'HOME' ENTERED AT 14:59:02 ON 05 DEC 2005

=> fil medline biosis caplus embase wpids

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.48

FILE 'MEDLINE' ENTERED AT 14:59:18 ON 05 DEC 2005

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=> (kinesin or microtubule) (s) inhibit?
L1 10995 (KINESIN OR MICROTUBULE) (S) INHIBIT?

=> (kinesin and microtubule) (s) inhibitor
PROXIMITY OPERATION NOT ALLOWED
Certain operators may not be nested in combination with other operators. A nested operator is valid only when it occurs at the same level or above the operator outside the nested phrase as determined by the following precedence list:

1. Numeric
2. (W), (NOTW), (A), (NOTA)
3. (S), (NOTS)
4. (P), (NOTP)
5. (L), (NOTL)
6. AND, NOT
7. OR

For example, '(MONOCLONAL(W)ANTIBOD?)(L)ANTIGEN?' is valid since (W) is above (L) on the precedence list. However, '((THIN(W)LAYER)(L)PHOSPHOLIPID#)(A)LACTONE#' is not valid since (L) is below (A) on the precedence list. The only exception is the 'OR' operator. This operator may be used in combination with any other operator. For example, '(ATOMIC OR NUCLEAR)(W)REACTOR' is valid.

=> end

ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:n

=> kinesin (s) microtubule (s) inhibitor
L2 19 KINESIN (S) MICROTUBULE (S) INHIBITOR

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3 10 DUP REM L2 (9 DUPLICATES REMOVED)

=> t ti l3 1-10

L3 ANSWER 1 OF 10 CAPLUS COPYRIGHT 2005 ACS on STN
TI External mechanical force as an inhibition process in kinesin's motion

L3 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2005 ACS on STN
TI Monastrol, a selective inhibitor of the mitotic kinesin Eg5, induces a distinctive growth profile of dendrites and axons in primary cortical neuron cultures

L3 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2005 ACS on STN
TI Interaction of the Mitotic Inhibitor Monastrol with Human Kinesin Eg5

L3 ANSWER 4 OF 10 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 1
TI Evidence for kinesin- and dynein-like protein function in circular nuclear migration in the green alga Pleurenterium tumidum: Digital time lapse analysis of inhibitor effects.

L3 ANSWER 5 OF 10 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Visualization of translated tau protein in the axons of neuronal P19 cells and characterization of tau RNP granules.

L3 ANSWER 6 OF 10 MEDLINE on STN DUPLICATE 2
TI Role of mitotic motors, dynein and kinesin, in the induction of abnormal centrosome integrity and multipolar spindles in cultured V79 cells exposed to dimethylarsinic acid.

L3 ANSWER 7 OF 10 MEDLINE on STN DUPLICATE 3
TI Microtubule-dependent assembly of the nuclear envelope in Xenopus laevis egg extract.

L3 ANSWER 8 OF 10 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 4
TI Adociasulfate 10, a new merohexaprenoid sulfate from the sponge Haliclona (aka Adocia) sp.

L3 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2005 ACS on STN
TI Inhibitors of Kinesin Activity from Structure-Based Computer Screening

L3 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2005 ACS on STN
TI A marine natural product inhibitor of Kinesin motors

=> d ibib abs l3 1-10

L3 ANSWER 1 OF 10 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2005:715529 CAPLUS
DOCUMENT NUMBER: 143:281133
TITLE: External mechanical force as an inhibition process in kinesin's motion
AUTHOR(S): Ciudad, Aleix; Sancho, Jose Maria
CORPORATE SOURCE: Departament d'Estructura i Constituents de la Materia,

Facultat de Fisica, Universitat de Barcelona, Barcelona, 08028, Spain
SOURCE: Biochemical Journal (2005), 390(1), 345-349
CODEN: BIJOAK; ISSN: 0264-6021
PUBLISHER: Portland Press Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We analyzed published force-velocity data for kinesin using classical Michaelis-Menten kinetic theory and found that the effect of force on the stepping rate of kinesin is analogous to the effect of a mixed inhibitor in classical inhibition theory. We derived an anal. expression for the velocity of kinesin (the stepping rate, equal to the ATP turnover rate) as a function of ATP concentration and force, and showed that it accurately predicts the observed single mol. stepping rate of kinesin under a variety of conditions.
REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2005:363423 CAPLUS
DOCUMENT NUMBER: 143:23604
TITLE: Monastrol, a selective inhibitor of the mitotic kinesin Eg5, induces a distinctive growth profile of dendrites and axons in primary cortical neuron cultures
AUTHOR(S): Yoon, Seung Yong; Choi, Jung Eun; Huh, Jae-Wan; Hwang, Onyou; Lee, Hui Sun; Hong, Hea Nam; Kim, Donghou
CORPORATE SOURCE: Department of Anatomy and Cell Biology, University of Ulsan College of Medicine, Seoul, S. Korea
SOURCE: Cell Motility and the Cytoskeleton (2005), 60(4), 181-190
CODEN: CMCYEO; ISSN: 0886-1544
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Various factors including some motor proteins regulate microtubule (MT) transport and influence the formation of neuronal processes. Eg5, a slow and non-processive (+)-end directed motor mol., is expressed in developing and differentiated neurons. However, how Eg5 works in neurons is still elusive. Thus, we treated primary rat cortical neuron cultures with monastrol, a specific inhibitor of Eg5, to investigate its role in neurons. Immature neurons treated with monastrol extended longer processes than control within a few hours. After 3 days, immature neurons treated with monastrol had longer dendrites but slightly shorter axons than control. This difference in growth between dendrites and axons became more prominent as the cells differentiated until 5 days. Interestingly, MT distributions in the cell bodies of monastrol-treated neurons appeared somewhat circular surrounding the nucleus, while MTs in the cell bodies of control neurons were primarily distributed in the MT organizing center (MTOC) just beside the nucleus. In mature neurons, monastrol treatment induced the axonal clusters of tubulins, grossly not affecting dendrites. Taken together, we conclude that Eg5 acts distinctively on dendrites and axons in neurons and suggest a putative model of how Eg5 works distinctively on dendrites and axons.
REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:954400 CAPLUS
DOCUMENT NUMBER: 138:149288
TITLE: Interaction of the Mitotic Inhibitor Monastrol with Human Kinesin Eg5

AUTHOR(S): DeBonis, Salvatore; Simorre, Jean-Pierre; Crevel, Isabelle; Lebeau, Luc; Skoufias, Dimitrios A.; Blangy, Anne; Ebel, Christine; Gans, Pierre; Cross, Robert; Hackney, David D.; Wade, Richard H.; Kozielski, Frank
CORPORATE SOURCE: Institut de Biologie Structurale, Grenoble, 38027, Fr.
SOURCE: Biochemistry (2003), 42(2), 338-349
CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The microtubule-dependent kinesin-like protein Eg5 from Homo sapiens is involved in the assembly of the mitotic spindle. It shows a three-domain structure with an N-terminal motor domain, a central coiled coil, and a C-terminal tail domain. In vivo HsEg5 is reversibly inhibited by monastrol, a small cell-permeable mol. that causes cells to be arrested in mitosis. Both monomeric and dimeric Eg5 constructs have been examined in order to define the minimal monastrol binding domain on HsEg5. NMR relaxation expts. show that monastrol interacts with all of the Eg5 constructs used in this study. Enzymic techniques indicate that monastrol partially inhibits Eg5 ATPase activity by binding directly to the motor domain. The binding is noncompetitive with respect to microtubules, indicating that monastrol does not interfere with the formation of the motor-MT complex. The binding is not competitive with respect to ATP. Both enzymol. and in vivo assays show that the S enantiomer of monastrol is more active than the R enantiomer and racemic monastrol. Stopped-flow fluorometry indicates that monastrol inhibits ADP release by forming an Eg5-ADP-monastrol ternary complex. Monastrol reversibly inhibits the motility of human Eg5. Monastrol has no inhibitory effect on the following members of the kinesin superfamily: MC5 (Drosophila melanogaster Ncd), HK379 (H. sapiens conventional kinesin), DKH392 (D. melanogaster conventional kinesin), BimC1-428 (Aspergillus nidulans BimC), Klp15 (Caenorhabditis elegans C-terminal motor), or Nkin460GST (Neurospora crassa conventional kinesin).

REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 10 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 1
ACCESSION NUMBER: 2003:179082 BIOSIS
DOCUMENT NUMBER: PREV200300179082
TITLE: Evidence for kinesin- and dynein-like protein function in circular nuclear migration in the green alga Pleurenterium tumidum: Digital time lapse analysis of inhibitor effects.
AUTHOR(S): Holzinger, Andreas; Luetz-Meindl, Ursula [Reprint Author]
CORPORATE SOURCE: Institute of Plant Physiology, University of Salzburg, A-5020, Salzburg, Austria
Ursula.Meindl@sbg.ac.at
SOURCE: Journal of Phycology, (February 2003) Vol. 39, No. 1, pp. 106-114. print.
ISSN: 0022-3646 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Apr 2003
Last Updated on STN: 9 Apr 2003

AB The unicellular green alga Pleurenterium tumidum Breb. performs a unique type of circular nuclear migration, wherein the nucleus leaves its central position and starts revolutions in the cortical isthmus area about 10 h after mitosis. This motion lasts for at least 12 h with an average velocity of about 1 h per revolution. Possible force generation modes during circular nuclear migration of Pleurenterium were investigated by application of inhibitors and the use of digital time-lapse video microscopy. 5'-Adenylylimidodiphosphate, a nonhydrolyzable nucleotide analogue, retarded or inhibited circular nuclear migration, suggesting

that ATPase dependent motor proteins are involved. Adociasulfate-2, a **kinesin** specific **inhibitor**, caused displacement of the nucleus, suggesting that the linkage between the **microtubule** track and the nucleus is lost. The nucleus was still able to move for short distances, but no normal revolutions took place. Erythro-9-(3-(2-hydroxynonyl)) adenine, a dynein ATPase inhibitor, led to complete inhibition of nuclear revolutions, suggesting a function in force generation also for this molecular motor. In addition, kinesin- and dynein-like proteins were detected in Pleurenterium extracts by Western blotting. The myosin specific inhibitor 2,3-butanedione 2-monoxime did not influence circular nuclear migration in Pleurenterium. This result and the absence of actin filaments around the migrating nucleus as depicted by means of microinjection of Alexa phalloidin in the present study indicate that the actin-myosin system can be excluded from force generation.

L3 ANSWER 5 OF 10 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:565171 BIOSIS
DOCUMENT NUMBER: PREV200200565171
TITLE: Visualization of translated tau protein in the axons of neuronal P19 cells and characterization of tau RNP granules.
AUTHOR(S): Aronov, Stella; Aranda, Gonzalo; Behar, Leah; Ginzburg, Irith [Reprint author]
CORPORATE SOURCE: Department of Neurobiology, The Weizmann Institute of Science, Rehovot, 76100, Israel
irith.ginzburg@weizmann.ac.il
SOURCE: Journal of Cell Science, (October 1, 2002) Vol. 115, No. 19, pp. 3817-3827. print.
CODEN: JNCSAI. ISSN: 0021-9533.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Nov 2002
Last Updated on STN: 7 Nov 2002

AB Localization of tau mRNA to the axon requires the axonal localization cis signal (ALS), which is located within the 3' untranslated region, and trans-acting binding proteins, which are part of the observed granular structures in neuronal cells. In this study, using both biochemical and morphological methods, we show that the granules contain tau mRNA, HuD RNA-binding protein, which stabilizes mRNA, and KIF3A, a member of the kinesin microtubule-associated motor protein family involved in anterograde transport. The granules are detected along the axon and accumulate in the growth cone. Inhibition of KIF3A expression caused neurite retraction and inhibited tau mRNA axonal targeting. Taken together, these results suggest that HuD and KIF3A proteins are present in the tau mRNA axonal granules and suggest an additional function for the **kinesin** motor family in the **microtubule**-dependent translocation of RNA granules Localized tau-GFP expression was blocked by a protein synthesis **inhibitor**, and upon release from inhibition, nascent tau-GFP 'hot spots' were directly observed in the axon and growth cones. These observations are consistent with local protein synthesis in the axon resulting from the transported tau mRNA.

L3 ANSWER 6 OF 10 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2002078902 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11804606
TITLE: Role of mitotic motors, dynein and kinesin, in the induction of abnormal centrosome integrity and multipolar spindles in cultured V79 cells exposed to dimethylarsinic acid.
AUTHOR: Ochi Takafumi
CORPORATE SOURCE: Department of Toxicology and Environmental Health, Faculty of Pharmaceutical Sciences, Teikyo University, Sagamiko,

Kanagawa 199-0195, Japan.. tkfmochi@pharm.teikyo-u.ac.jp
SOURCE: Mutation research, (2002 Jan 29) 499 (1) 73-84.
Journal code: 0400763. ISSN: 0027-5107.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 20020128
Last Updated on STN: 20020320
Entered Medline: 20020319

AB The role of **microtubule**-based motors in the induction of abnormal centrosome integrity by dimethylarsinic acid (DMAA) was investigated with the use of monastrol, a specific **inhibitor** of mitotic **kinesin**, and vanadate, an **inhibitor** of dynein ATPase. Cytoplasmic dynein co-localized with multiple foci of gamma-tubulin in mitotic cells arrested by DMAA. Disruption of microtubules caused dispersion of dynein while multiple foci of gamma-tubulin were coalesced to a single dot. Vanadate also caused dispersion of dynein, which had been co-localized with multiple foci of gamma-tubulin by DMAA, without affecting spindle organization. However, the dispersion of dynein did not prohibit the induction of abnormal centrosome integrity by DMAA. Inhibition of mitotic kinesin by monastrol resulted in monoastral cells with non-migrated centrosomes in the cell center. Monastrol, when applied to mitotic cells with abnormal centrosome integrity, rapidly reduced the incidence of cells with the centrosome abnormality. Moreover, monastrol completely inhibited reorganization of abnormal centrosomes that had been coalesced to a single dot by microtubule disruption. These results suggest that abnormal centrosome integrity caused by DMAA is not simply due to dispersion of fragments of microtubule-organizing centers, but is dependent on the action of kinesin. In addition, the results suggest that kinesin plays a role not only in the induction of mitotic centrosome abnormality, but also in maintenance.

L3 ANSWER 7 OF 10 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2002095682 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11824787
TITLE: Microtubule-dependent assembly of the nuclear envelope in Xenopus laevis egg extract.
AUTHOR: Ewald A; Zunkler C; Lourim D; Dabauvalle M C
CORPORATE SOURCE: Department of Cell and Developmental Biology, Theodor-Boveri-Institute, University of Wurzburg, Germany.
SOURCE: European journal of cell biology, (2001 Nov) 80 (11) 678-91.
Journal code: 7906240. ISSN: 0171-9335.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200207
ENTRY DATE: Entered STN: 20020205
Last Updated on STN: 20020717
Entered Medline: 20020716

AB Microtubules take part in several mechanisms of intracellular motility, including organelle transport and mitosis. We have studied the ability of Xenopus egg extract to support nuclear membrane and pore complex formation when microtubule dynamics are manipulated. In this report we show that the formation of a nuclear envelope surrounding sperm chromatin requires polymerized microtubules. We have observed that **microtubule** -depolymerizing reagents, and AS-2, a known **inhibitor** of the **microtubule** motor protein **kinesin**, do not inhibit the formation of a double nuclear membrane. However these double membranes contain no morphologically identifiable nuclear pore complexes and do not

support the accumulation of karyophilic proteins. In contrast, the assembly of annulate lamellae, cytoplasmic structures containing a subset of pore complex proteins, was not affected. Our data show that not only polymerized microtubules, but also the microtubule motor protein kinesin, are involved in the formation of the nuclear envelope. These results support the conclusion that multiple nuclear envelope-forming mitotic vesicle populations exist, that microtubules play an essential and selective role in the transport of nuclear envelope-forming vesicle population(s), and that separate mechanisms are involved in nuclear envelope and annulate lamellae formation.

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DUPLICATE 4
ACCESSION NUMBER: 2000:543998 BIOSIS
DOCUMENT NUMBER: PREV200000543998
TITLE: Adociasulfate 10, a new merohexaprenoid sulfate from the sponge Haliclona (aka Adocia) sp.
AUTHOR(S): Blackburn, Christine L.; Faulkner, D. John [Reprint author]
CORPORATE SOURCE: Marine Research Division, Scripps Institution of Oceanography, University of California at San Diego, La Jolla, CA, 92093-0212: jfaulkner@ucsd.edu, USA
SOURCE: Tetrahedron, (20 October, 2000) Vol. 56, No. 43, pp. 8429-8432. print.
CODEN: TETRAB. ISSN: 0040-4020.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 13 Dec 2000
Last Updated on STN: 11 Jan 2002
AB The meroterpenoid adociasulfate 10 was isolated from the Palauan sponge Haliclona (aka Adocia) sp., together with the previously reported metabolites adociasulfates 1-6. Unlike other adociasulfates, which contain mono- or di-sulfated hydroquinones, adociasulfate 10 contains an unusual glycolic acid residue in place of one of the sulfate groups. Adociasulfate 10 is an **inhibitor** of the **kinesin** family of **microtubule** motor proteins with an IC50 of 7 muM.

L3 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2000:114711 CAPLUS
DOCUMENT NUMBER: 132:290326
TITLE: Inhibitors of Kinesin Activity from Structure-Based Computer Screening
AUTHOR(S): Hopkins, Seth C.; Vale, Ronald D.; Kuntz, Irwin D.
CORPORATE SOURCE: Department of Pharmaceutical Chemistry and Department of Cellular and Molecular Pharmacology, Howard Hughes Medical Institute University of California, San Francisco, CA, 94143-0446, USA
SOURCE: Biochemistry (2000), 39(10), 2805-2814
CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Kinesin motor proteins use ATP hydrolysis for transport along microtubules in the cell. We sought to identify small organic ligands that inhibit the activity of kinesin. Candidate mols. were identified by computational docking of com. available compds. using the computer program DOCK. Compds. were docked at either of two sites, and a selection was tested for inhibition of microtubule-stimulated ATPase activity. Twenty-two submillimolar inhibitors were identified. Several inhibitors appeared to be competitive for microtubule binding and not for ATP binding, and three compds. showed 50% inhibition down to single-digit micromolar levels. Most inhibitors grouped into four distinct classes (fluoresceins, phenolphthaleins, anthraquinones, and naphthylene sulfonates). We measured the binding of the inhibitor rose bengal lactone (RBL) to kinesin

(dissociation constant 2.5 μM) by its increase in steady-state fluorescence anisotropy. The RBL binding site on kinesin was localized by fluorescent resonance energy transfer (FRET) using a donor fluorophore (coumarin) covalently attached at unique, surface-exposed cysteine residues engineered at positions 28, 149, 103, 220, or 330. RBL was found to bind in its original docked site, which is the pocket cradled by loop 8 and β-strand 5 in kinesin's three-dimensional structure. These results confirm the role of this region in microtubule binding and identify this pocket as a novel binding site for kinesin inhibition.
REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1998:260888 CAPLUS
DOCUMENT NUMBER: 129:37823
TITLE: A marine natural product inhibitor of Kinesin motors
AUTHOR(S): Sakowicz, Roman; Berdelis, Michael S.; Ray, Krishanu; Blackburn, Christine L.; Hopmann, Cordula; Faulkner, D. John; Goldstein, Lawrence S. B.
CORPORATE SOURCE: Dep. Pharmacoloy, Div. Cellular and Molecular Med., Howard Hughes Med. Inst., Univ. California, La Jolla, CA, 92093-0683, USA
SOURCE: Science (Washington, D. C.) (1998), 280(5361), 292-295
CODEN: SCIEAS; ISSN: 0036-8075
PUBLISHER: American Association for the Advancement of Science
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Members of the kinesin superfamily of motor proteins are essential for mitotic and meiotic spindle organization, chromosome segregation, organelle and vesicle transport, and many other processes that require microtubule-based transport. A compound, adociasulfate-2, was isolated from a marine sponge, Haliclona (also known as Adocia) species, that inhibited kinesin activity by targeting its motor domain and mimicking the activity of the microtubule. Thus, the kinesin-microtubule interaction site could be a useful target for small mol. modulators, and adociasulfate-2 should serve as an archetype for specific inhibitors of kinesin functions.
REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	41.61	42.09

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-3.65	-3.65

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	1.02	43.11

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL

	ENTRY	SESSION
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FILE 'MEDLINE' ENTERED AT 15:23:47 ON 05 DEC 2005

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=> Vale Ronald?/au
L4          305 VALE RONALD?/AU

=> kinesin and l4
L5          171 KINESIN AND L4

=> inhibitor and l5
L6            2 INHIBITOR AND L5

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7            1 DUP REM L6 (1 DUPLICATE REMOVED)
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=> d ibib abs l7

L7 ANSWER 1 OF 1 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 1
ACCESSION NUMBER: 2000:190314 BIOSIS
DOCUMENT NUMBER: PREV200000190314
TITLE: Inhibitors of **kinesin** activity from structure-based computer screening.
AUTHOR(S): Hopkins, Seth C. [Reprint author]; **Vale, Ronald D.** ; Kuntz, Irwin D.
CORPORATE SOURCE: Sepracor, 111 Locke Dr., Marlborough, MA, 01752, USA
SOURCE: Biochemistry, (March 14, 2000) Vol. 39, No. 10, pp. 2805-2814. print.
CODEN: BICHAW. ISSN: 0006-2960.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 17 May 2000
Last Updated on STN: 4 Jan 2002

AB **Kinesin** motor proteins use ATP hydrolysis for transport along microtubules in the cell. We sought to identify small organic ligands to inhibit **kinesin**'s activity. Candidate molecules were identified by computational docking of commercially available compounds using the computer program DOCK. Compounds were docked at either of two sites, and a selection was tested for inhibition of microtubule-stimulated ATPase activity. Twenty-two submillimolar inhibitors were identified. Several inhibitors appeared to be competitive for microtubule binding and not for ATP binding, and three compounds showed 50% inhibition down to single-digit micromolar levels. Most inhibitors grouped into four distinct classes (fluoresceins, phenolphthaleins, anthraquinones, and naphthylene sulfonates). We measured the binding of one **inhibitor** , rose bengal lactone (RBL), to **kinesin** (dissociation constant 2.5 muM) by its increase in steady-state fluorescence anisotropy. The RBL binding site on **kinesin** was localized by fluorescent resonance energy transfer (FRET) using a donor fluorophore (coumarin) covalently attached at unique, surface-exposed cysteine residues engineered at positions 28, 149, 103, 220, or 330. RBL was found to bind in its original docked site: the pocket cradled by loop 8 and beta-strand 5 in **kinesin**'s three-dimensional structure. These results confirm this region's role in microtubule binding and identify this pocket as a novel binding site for **kinesin** inhibition.

=> dup rem l5
PROCESSING COMPLETED FOR L5
L8 108 DUP REM L5 (63 DUPLICATES REMOVED)

=> inhib? and l8
L9 10 INHIB? AND L8

=> l9 not l6
L10 9 L9 NOT L6

=> t ti l10 1-9

L10 ANSWER 1 OF 9 MEDLINE on STN
TI Two mitotic kinesins cooperate to drive sister chromatid separation during anaphase.

L10 ANSWER 2 OF 9 MEDLINE on STN
TI The roles of microtubule-based motor proteins in mitosis: comprehensive RNAi analysis in the Drosophila S2 cell line.

L10 ANSWER 3 OF 9 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Rapid movements of vimentin on microtubule tracks: **Kinesin** -dependent assembly of intermediate filament networks.

L10 ANSWER 4 OF 9 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Chemomechanical cycle of **kinesin** differs from that of myosin.

L10 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN
TI Single-molecule analysis of **kinesin** motility reveals regulation by the cargo-binding tail domain

L10 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN
TI Suppression of **kinesin** expression in cultured hippocampal neurons using antisense oligonucleotides

L10 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN
TI Identification of a **kinesin**-like microtubule-based motor protein in Dictyostelium discoideum

L10 ANSWER 8 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN
TI Characterization of the microtubule movement produced by sea urchin egg **kinesin**

L10 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN
TI Different axoplasmic proteins generate movement in opposite directions along microtubules in vitro

=> d ibib abs l10 1-9

L10 ANSWER 1 OF 9 MEDLINE on STN
ACCESSION NUMBER: 2004037668 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14681690
TITLE: Two mitotic kinesins cooperate to drive sister chromatid separation during anaphase.
COMMENT: Comment in: Nature. 2004 Jan 22;427(6972):300-1. PubMed ID: 14737150
AUTHOR: Rogers Gregory C; Rogers Stephen L; Schwimmer Tamara A; Ems-McClung Stephanie C; Walczak Claire E; **Vale Ronald D**; Scholey Jonathan M; Sharp David J
CORPORATE SOURCE: Department of Physiology and Biophysics, Albert Einstein College of Medicine, Bronx, New York 10461, USA.
SOURCE: Nature, (2004 Jan 22) 427 (6972) 364-70. Electronic Publication: 2003-12-14.
Journal code: 0410462. ISSN: 1476-4687.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200402
ENTRY DATE: Entered STN: 20040123
Last Updated on STN: 20040212
Entered Medline: 20040211
AB During anaphase identical sister chromatids separate and move towards opposite poles of the mitotic spindle. In the spindle, kinetochore microtubules have their plus ends embedded in the kinetochore and their minus ends at the spindle pole. Two models have been proposed to account for the movement of chromatids during anaphase. In the 'Pac-Man' model, kinetochores induce the depolymerization of kinetochore microtubules at their plus ends, which allows chromatids to move towards the pole by 'chewing up' microtubule tracks. In the 'poleward flux' model,

kinetochores anchor kinetochore microtubules and chromatids are pulled towards the poles through the depolymerization of kinetochore microtubules at the minus ends. Here, we show that two functionally distinct microtubule-destabilizing KinI **kinesin** enzymes (so named because they possess a **kinesin**-like ATPase domain positioned internally within the polypeptide) are responsible for normal chromatid-to-pole motion in Drosophila. One of them, KLP59C, is required to depolymerize kinetochore microtubules at their kinetochore-associated plus ends, thereby contributing to chromatid motility through a Pac-Man-based mechanism. The other, KLP10A, is required to depolymerize microtubules at their pole-associated minus ends, thereby moving chromatids by means of poleward flux.

L10 ANSWER 2 OF 9 MEDLINE on STN
ACCESSION NUMBER: 2003434334 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12975346
TITLE: The roles of microtubule-based motor proteins in mitosis: comprehensive RNAi analysis in the Drosophila S2 cell line.
AUTHOR: Goshima Gohta; **Vale Ronald D**
CORPORATE SOURCE: Department of Cellular and Molecular Pharmacology, University of California, San Francisco, San Francisco, CA 94107, USA.
SOURCE: Journal of cell biology, (2003 Sep 15) 162 (6) 1003-16. Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200310
ENTRY DATE: Entered STN: 20030917
Last Updated on STN: 20031101
Entered Medline: 20031031

AB Kinesins and dyneins play important roles during cell division. Using RNA interference (RNAi) to deplete individual (or combinations of) motors followed by immunofluorescence and time-lapse microscopy, we have examined the mitotic functions of cytoplasmic dynein and all 25 kinesins in Drosophila S2 cells. We show that four kinesins are involved in bipolar spindle assembly, four kinesins are involved in metaphase chromosome alignment, dynein plays a role in the metaphase-to-anaphase transition, and one **kinesin** is needed for cytokinesis. Functional redundancy and alternative pathways for completing mitosis were observed for many single RNAi knockdowns, and failure to complete mitosis was observed for only three kinesins. As an example, **inhibition** of two microtubule-depolymerizing kinesins initially produced monopolar spindles with abnormally long microtubules, but cells eventually formed bipolar spindles by an acentrosomal pole-focusing mechanism. From our phenotypic data, we construct a model for the distinct roles of molecular motors during mitosis in a single metazoan cell type.

L10 ANSWER 3 OF 9 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:492856 BIOSIS
DOCUMENT NUMBER: PREV199800492856
TITLE: Rapid movements of vimentin on microtubule tracks: **Kinesin**-dependent assembly of intermediate filament networks.
AUTHOR(S): Prahlad, Veena; Yoon, Miri; Moair, Robert D.; **Vale, Ronald D.**; Goldman, Robert D. [Reprint author]
CORPORATE SOURCE: Dep. Cell Mol. Biol., Northwestern Univ. Medical Sch., 303 E. Chicago Ave., Chicago, IL 60611, USA
SOURCE: Journal of Cell Biology, (Oct. 5, 1998) Vol. 143, No. 1, pp. 159-170. print.
CODEN: JCLBA3. ISSN: 0021-9525.

DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 18 Nov 1998
Last Updated on STN: 18 Nov 1998

AB The assembly and maintenance of an extended intermediate filament (IF) network in fibroblasts requires microtubule (MT) integrity, Using a green fluorescent protein-vimentin construct, and spreading BHK-21 cells as a model system to study IF-MT interactions, we have discovered a novel mechanism involved in the assembly of the vimentin IF cytoskeleton. This entails the rapid, discontinuous, and MT-dependent movement of IF precursors towards the peripheral regions of the cytoplasm where they appear to assemble into short fibrils. These precursors, or vimentin dots, move at speeds averaging 0.55 +- 0.24 mum/s. The vimentin dots colocalize with MT and their motility is **inhibited** after treatment with nocodazole. Our studies further implicate a conventional **kinesin** in the movement of the vimentin dots. The dots colocalize with conventional **kinesin** as shown by indirect immunofluorescence, and IF preparations from spreading cells are enriched in **kinesin**. Furthermore, microinjection of **kinesin** antibodies into spreading cells prevents the assembly of an extended IF network. These studies provide insights into the interactions between the IF and MT systems. They also suggest a role for conventional **kinesin** in the distribution of non-membranous protein cargo, and the local regulation of IF assembly.

L10 ANSWER 4 OF 9 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1993:166167 BIOSIS
DOCUMENT NUMBER: PREV199395087217
TITLE: Chemomechanical cycle of **kinesin** differs from that of myosin.
AUTHOR(S): Romberg, Laura; **Vale, Ronald D.** [Reprint author]
CORPORATE SOURCE: Dep. Pharmacol., Univ. Calif., San Francisco, CA 94143, USA
SOURCE: Nature (London), (1993) Vol. 361, No. 6408, pp. 168-170.
CODEN: NATUAS. ISSN: 0028-0836.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 31 Mar 1993
Last Updated on STN: 1 Apr 1993

AB Motor proteins move unidirectionally along cytoskeletal polymers by coupling translocation to cycles of ATP hydrolysis. The energy from ATP is required both to generate force and to dissociate the motor-filament complex in order to begin a new chemomechanical cycle. For myosin, force production is associated with phosphate release following ATP hydrolysis, whereas dissociation of actomyosin is tightly coupled to the binding of ATP. Dynein, a microtubule motor, uses a similar cycle, suggesting that all cytoskeletal motor might operate by a common mechanism. Here we investigate kinensin's chemomechanical cycle by assaying microtubule movement by single **kinesin** molecules when intermediate states in the hydrolysis cycle are prolonged with ATP analogues or **inhibitors**. In contrast to myosin and dynein, **kinesin** with bound ADP dissociates from microtubules during translocation, whereas **kinesin** with unhydrolyzed nucleotide remains tightly associated with the polymer. These findings imply that **kinesin** converts ATP energy into mechanical work by a pathway distinct from that of myosin or dynein.

L10 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1999:594297 CAPLUS
DOCUMENT NUMBER: 131:319198
TITLE: Single-molecule analysis of **kinesin** motility reveals regulation by the cargo-binding tail domain
AUTHOR(S): Friedman, Dara S.; **Vale, Ronald D.**

CORPORATE SOURCE: Department of Cellular and Molecular Pharmacology, University of California, San Francisco, CA, 94143, USA
SOURCE: Nature Cell Biology (1999), 1(5), 293-297
CODEN: NCBIFN; ISSN: 1465-7392
PUBLISHER: Macmillan Magazines Ltd
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Conventional **kinesin** transports membranes along microtubules in vivo, but the majority of cellular **kinesin** is unattached to cargo. The motility of non-cargo-bound, soluble **kinesin** may be repressed by an interaction between the amino-terminal motor and carboxy-terminal cargo-binding tail domains, but neither bead nor microtubule-gliding assays have shown such **inhibition**. Here we use a single-mol. assay that measures the motility of **kinesin** unattached to a surface. We show that full-length **kinesin** binds microtubules and moves about ten times less frequently and exhibits discontinuous motion compared with a truncated **kinesin** lacking a tail. Mutation of either the stalk hinge or neck coiled-coil domain activates motility of full-length **kinesin**, indicating that these regions are important for tail-mediated repression. Our results suggest that the motility of soluble **kinesin** in the cell is **inhibited** and that the motor becomes activated by cargo binding.

REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1992:211877 CAPLUS
DOCUMENT NUMBER: 116:211877
TITLE: Suppression of **kinesin** expression in cultured hippocampal neurons using antisense oligonucleotides
AUTHOR(S): Ferreira, Adriana; Niclas, Joshua; **Vale, Ronald D.**; Banker, Gary; Kosik, Kenneth S.
CORPORATE SOURCE: Sch. Med., Univ. Virginia, Charlottesville, VA, 22908, USA
SOURCE: Journal of Cell Biology (1992), 117(3), 595-606
CODEN: JCLBA3; ISSN: 0021-9525
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Kinesin**, a microtubule-based force-generating mol., is thought to translocate organelles along microtubules. To examine the function of **kinesin** in neurons, it was sought to suppress **kinesin** heavy chain (KHC) expression in cultured hippocampal neurons using antisense oligonucleotides and study the phenotype of these KHC null cells. Two different antisense oligonucleotides complementary to the KHC sequence reduced the protein levels of the heavy chain by >95% within 24 h after application and produced identical phenotypes. After **inhibition** of KHC expression for 24 or 48 h, neurons extended an array of neurites often with 1 neurite longer than the others; however, the length of all these neurites was significantly reduced. **Inhibition** of KHC expression also altered the distribution of GAP-43 and synapsin I, 2 proteins thought to be transported in association with membranous organelles. These proteins, which are normally localized at the tips of growing neurites, were confined to the cell body in antisense-treated cells. Treatment of the cells with the corresponding sense oligonucleotides affected neither the distribution of GAP-43 and synapsin I, nor the length of neurites. A full recovery of neurite length occurred after removal of the antisense oligonucleotides from the medium. These data indicate that KHC plays a role in the anterograde translocation of vesicles containing GAP-43 and synapsin I. A deficiency in vesicle delivery may also explain the **inhibition** of neurite outgrowth.

Despite the **inhibition** of KHC and the failure of GAP-43 and synapsin I to move out of the cell body, hippocampal neurons can extend processes and acquire an asym. morphol.

L10 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1990:18630 CAPLUS
DOCUMENT NUMBER: 112:18630
TITLE: Identification of a **kinesin**-like microtubule-based motor protein in Dictyostelium discoideum
AUTHOR(S): McCaffrey, Gretchen; **Vale, Ronald D.**
CORPORATE SOURCE: Dep. Pharmacol., Univ. California, San Francisco, CA, 94143, USA
SOURCE: EMBO Journal (1989), 8(11), 3229-34
CODEN: EMJODG; ISSN: 0261-4189
DOCUMENT TYPE: Journal
LANGUAGE: English
AB D. discoideum, a unicellular eukaryote amenable to both biochem. and genetic dissection, provides an attractive system for studying microtubule-based transport. In this work, microtubule-based motor activities were identified in Dictyostelium cell exts. and a protein was partially purified that induces microtubule translocation along glass surfaces. This protein, which sediments at .apprx.9S in sucrose d. gradients and is composed of a 105 kd polypeptide, generates anterograde movement along microtubules that is insensitive to 5 mM N-methylmaleimide but sensitive to 200 μM vanadate and has similar nucleotide-dependent microtubule binding properties to those of kinesins purified from mammals, sea urchins, and Drosophila. This **kinesin**-like mol. from Dictyostelium, however, is immunol. distinct from bovine and squid neuronal kinesins and supports microtubule movement on glass at 4-fold greater velocities (2.0 vs. 0.5 μm/s). AMP-PNP (adenylyl imidodiphosphate), which promotes attachment of previously characterized kinesins to microtubules, decreases the affinity of the Dictyostelium **kinesin** homolog for microtubules. Thus, an AMP-PNP-induced rigor binding may not be a characteristic of kinesins from lower eukaryotes.

L10 ANSWER 8 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1987:115462 CAPLUS
DOCUMENT NUMBER: 106:115462
TITLE: Characterization of the microtubule movement produced by sea urchin egg **kinesin**
AUTHOR(S): Porter, Mary E.; Scholey, Jonathan M.; Stemple, Derek L.; Vigers, Guy P. A.; **Vale, Ronald D.**; Sheetz, Michael P.; McIntosh, J. Richard
CORPORATE SOURCE: Dep. Mol., Cell. Dev. Biol., Univ. Colorado, Boulder, CO, 80309-0347, USA
SOURCE: Journal of Biological Chemistry (1987), 262(6), 2794-802
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
AB An in vitro assay was used to characterize some of the motile properties of sea urchin egg **kinesin** (I). I was purified via 5'-adenylyl imidodiphosphate-induced binding to taxol-assembled microtubules, extraction from the microtubules in ATP, and gel filtration chromatog. Partially purified I was then adsorbed to a glass coverslip, mixed with microtubules and ATP, and viewed by video-enhanced differential interference contrast microscopy. The microtubule translocating activity of purified egg I was qual. similar to the analogous activity observed in crude exts. of sea urchin eggs and resembled the activity of neuronal I with respect to both the maximal rate (>0.5 μm/s) and the direction of movement. Axonemes glided on a I-coated coverslip toward their minus ends, and I-coated beads

translocated toward the plus ends of centrosome microtubules. Sea urchin egg I was **inhibited** by high concns. of SH-group reagents. The nucleotide requirement of sea urchin egg I was fairly broad (ATP > GTP > ITP), and the rate of microtubule movement increased in a saturable fashion with the ATP concentration Thus, the motile activity of egg I was indistinguishable from that of neuronal I. Egg I may be associated with microtubule-based motility in vivo.

L10 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1986:83877 CAPLUS
DOCUMENT NUMBER: 104:83877
TITLE: Different axoplasmic proteins generate movement in opposite directions along microtubules in vitro
AUTHOR(S): **Vale, Ronald D.**; Schnapp, Bruce J.; Mitchison, Tim; Steuer, Eric; Reese, Thomas S.; Sheetz, Michael P.
CORPORATE SOURCE: Lab. Neurobiol., NINCDS, Woods Hole, MA, 02543, USA
SOURCE: Cell (Cambridge, MA, United States) (1985), 43(3, Pt. 2), 623-32
CODEN: CELLB5; ISSN: 0092-8674
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Single microtubules from squid axoplasm support bidirectional movement of organelles. A previously purified microtubule translocator (**kinesin**) moves beads in only 1 direction along microtubules. A polar array of microtubules assembled off of centrosomes in vitro was used to demonstrate that **kinesin** moves latex beads from the minus to the plus ends of microtubules, a direction that corresponds to anterograde transport in the axon. A crude solubilized fraction from squid axoplasm (S1a), however, generates bidirectional movement of beads along microtubules. Retrograde bead movement (1.4 μm/s) is **inhibited** by N-ethylmaleimide and 20 μM vanadate, whereas anterograde movement (0.6 μm/s) is unaffected by these agents. Furthermore, a monoclonal antibody against **kinesin**, when coupled to Sepharose, removes the anterograde, but not the retrograde, bead translocator from S1a. Thus, there is a retrograde bead translocator which is pharmacol. and immunol. distinct from **kinesin**.

=> d his

(FILE 'HOME' ENTERED AT 14:58:48 ON 05 DEC 2005)

FILE 'STNGUIDE' ENTERED AT 14:58:58 ON 05 DEC 2005

FILE 'HOME' ENTERED AT 14:59:02 ON 05 DEC 2005

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, WPIDS' ENTERED AT 14:59:18 ON 05 DEC 2005
L1 10995 (KINESIN OR MICROTUBULE) (S) INHIBIT?
L2 19 KINESIN (S) MICROTUBULE (S) INHIBITOR
L3 10 DUP REM L2 (9 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 15:04:44 ON 05 DEC 2005

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, WPIDS' ENTERED AT 15:23:47 ON 05 DEC 2005
L4 305 VALE RONALD?/AU
L5 171 KINESIN AND L4
L6 2 INHIBITOR AND L5
L7 1 DUP REM L6 (1 DUPLICATE REMOVED)
L8 108 DUP REM L5 (63 DUPLICATES REMOVED)

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L9            10 INHIB? AND L8
L10            9 L9 NOT L6

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COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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FULL ESTIMATED COST                                    54.81      97.98

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                      ENTRY    SESSION
CA SUBSCRIBER PRICE                                    -3.65      -7.30

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